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A REVERSED ACTION OF THE VAGUS ON THE MAMMALIAN HEART.

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Introductory. A cardio-accelerator action of the mammalian vagus has frequently been described, and in several instances has been regarded as indicating the presence in the vagus itself of true accelerator fibres. The earlier observations, such as those of Rutherford (1) (who, indeed, explained the phenomenon otherwise), of Boehm (2), or of Schiff (3), and the later experiments of Arloing (4), whether the accelerator action was revealed by the aid of drugs such as atropine or curare, or by degenerative section of the nerve, are alike open to explanation by the existence in the vagus of recurrent sympathetic fibres, arising from the stellate or the inferior cervical ganglion, joining the vagus by one of the communicating branches which unite it with the anterior limbs of the annulus, and looping back to their distribution in the cardiac branches. We have ourselves in one case, which will be mentioned later in more detail, obtained a result by means of degenerative section similar to those of Arloing, and susceptible of the same explanation. The phenomenon which is our main concern in this paper is of a different kind. It can be reproduced with regularity under given conditions, and we shall give evidence that it has no connexion with the true sympathetic system. Whether it is due to the presence in the vagus of fibres which are normally accelerator in function is still open to question.

[1] The phenomenon with which this paper deals was first observed accidentally by Dale and Symons in 1907 and partly investigated: after an interval the investigation was resumed and brought to its present stage by Dale and Laidlaw. For preliminary note see This *Journal*, xxxix. *Proc. Phys. Soc.* p. xiii. 1909.

The production of the reversed action.

For the production of the phenomenon in its most characteristic form certain conditions are necessary. The animal which exhibits it best is the cat. In one experiment on a ferret we obtained it without difficulty. In the dog it is seen, if at all, in a very weak form. In the rabbit we have never succeeded in obtaining the effect. The choice of an anæsthetic is not a matter of indifference. In our earlier experiments we used urethane, with varying success as regards the production of the phenomenon. Latterly we have found that the use of paraldehyde, which has little depressant action on the heart, enabled us to obtain uniformly successful results. The cats were anæsthetised in the first place with chloroform and ether: under these a tracheal cannula was inserted, and 1·5 c.c. of paraldehyde was then given by a stomach tube, the œsophagus being tied as a precaution against regurgitation. The administration of ether was gradually reduced as the effect of the paraldehyde became established. In the later stages of the experiment ether was usually unnecessary, since deep anæsthesia was maintained by the paraldehyde alone.

Blood-pressure was recorded by a mercury manometer connected with the left carotid artery. For most purposes the manometer movements were used as a record of the heart-beats, but in a few experiments the volume changes of the auricles and ventricles were separately observed by the use of the double cardiometer described by Lehndorff[5], small Brodie's bellows being used as recording instruments.

Either vagus produces the phenomenon equally well, and stimulation may be made with equally good result at any point from the ganglion of the trunk to the separation of the cardiac branches. The right vagus was usually chosen, simply as a matter of convenience, the artery cannula being in the left carotid. In a cat anæsthetised and prepared as above a preliminary stimulation of the vagus produced well-marked inhibition of the heart with the secondary coil at distances from the primary varying from 25—15 cm. in different animals. To produce the reversal phenomenon one of several drugs may be used. We observed it first with tropine, injected intravenously in doses of 50—150 mgm. Latterly we have used nicotine in intravenous doses of 1—2 mgm., with equally good results. Hordenine methiodide, the

action of which, as shown by one of us, is in most respects closely similar to that of nicotine, may be substituted in doses of 10—20 mgm., but the effect is more evanescent. Curare will also produce the phenomenon, though in less striking form. We will for convenience take as a type for description an experiment in which nicotine is used.

After the preliminary stimulation, for observation of the normal action of the unpoisoned vagus, 1 mgm. of nicotine is injected intravenously. The rise of blood-pressure resulting is allowed to pass off, when it will be found that the heart-beat is somewhat slower than before the nicotine was given. The vagus is again stimulated. If no inhibition is produced during stimulation the secondary coil is moved about 2 cm. nearer to the primary and the stimulus repeated. If inhibition now occurs during the stimulus another dose of 1 mgm. of nicotine is injected. Stimulation of the vagus will now produce either no inhibition during the stimulus, or a mere preliminary slowing for one or two beats, succeeded by a return to the original rate or, in some cases, a slight acceleration beyond it. When the stimulus ceases the rate gradually becomes slower. If no further stimulus is given this after-slowing, or delayed inhibition, gradually passes off and the original rate is regained. If, however, the stimulus be renewed, during the period of slow beating succeeding the first stimulus, the beat, with scarcely noticeable latent period, is rapidly accelerated up to or beyond the initial rate. The faster rate is maintained as long as stimulation is continued, and a further period of more marked slowing appears when the stimulation again ceases. If this process be repeated, the stimulation being in each case renewed before the slowing succeeding the previous

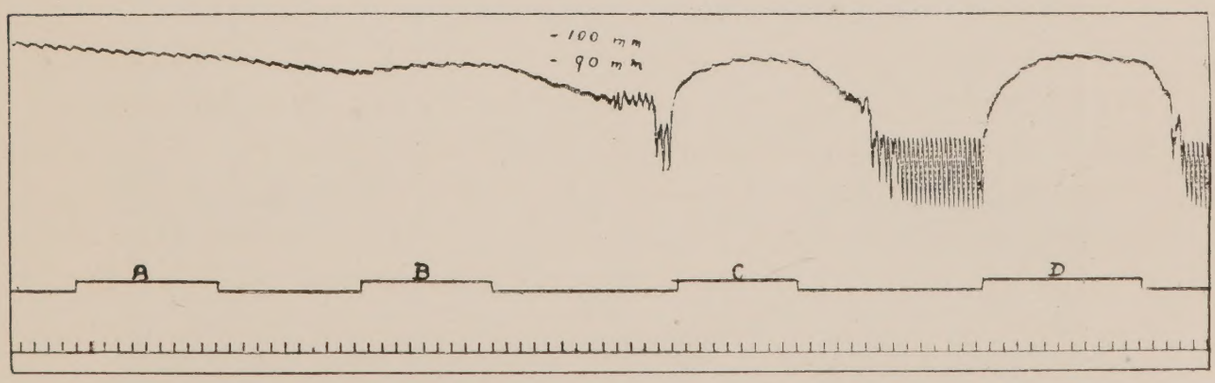

Fig. 1. [In this and other reproductions of kymographic tracings the lines of tracing, from above downwards, unless otherwise mentioned, are carotid blood-pressure (mercury manometer), signal key, and clock marking intervals of 2 seconds.]

Cat. Paraldehyde. Effect of four successive stimulations of the vagus after 1 mgm. of nicotine. At A and B coil at 20 cm.; at C and D coil at 18 cm.

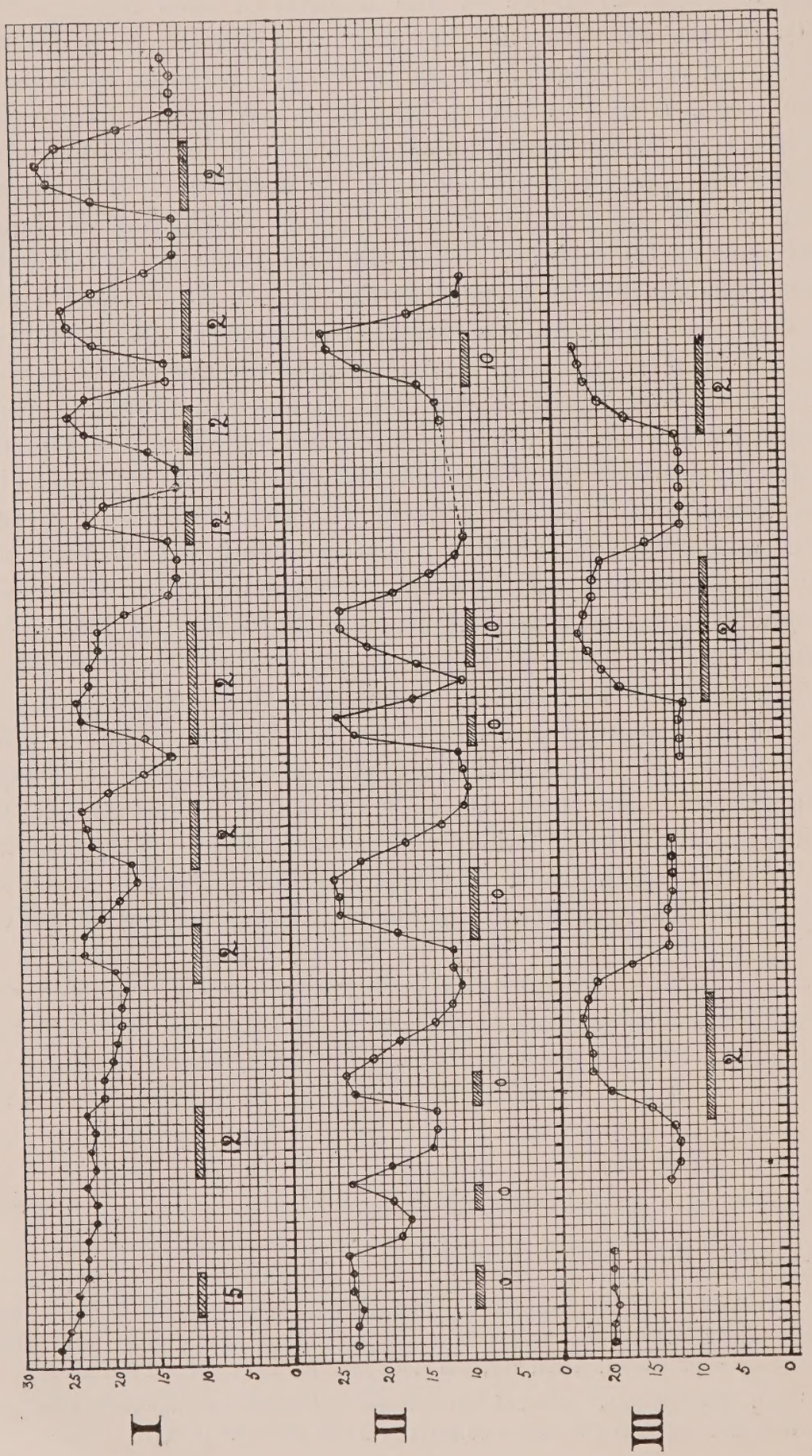

Fig. 2.

Fig. 2. Chart plotted from kymograph records of three experiments. Along the abscissæ time-intervals of 6 seconds are shown: the ordinates indicate the number of heart-beats in such intervals. The shaded rectangular areas indicate periods of vagus stimulation, the figures below them showing coil-distance in centimetres.

I. Development of the reversal phenomenon after 2 mgm. of nicotine.

II. ,, ,, ,, ,, ,, 100 mgm. of tropine.

III. Two longer periods of vagus stimulation from another experiment, in which complete reversal had been developed after 100 mgm. of tropine.

period of stimulation has passed off, the effect gradually becomes intensified, till after four or five such periods of stimulation the appearance is produced of marked inhibition *between* the stimuli, acceleration *during* the stimuli—the picture being almost the exact reverse of that produced by successive stimulations of the vagus in the normal animal.

Fig. 1 illustrates the onset and development of the phenomenon after 1 mgm. of nicotine. The chart (Fig. 2) was constructed from kymograph records by plotting as ordinates the number of heart-beats in consecutive periods of six seconds. It shows the development of the phenomenon after 100 mgm. of tropine (I) and after 2 mgm. of nicotine (II). The effects of two more prolonged periods of stimulation of the vagus, after the phenomenon was fully developed, are plotted in curve (III).

It has already been stated that the two vagi will give the effect equally well. They may be stimulated alternately with an effect not differing from that produced by repeated stimulation of one only. For example, stimulation of the left vagus, during the period of slow beating

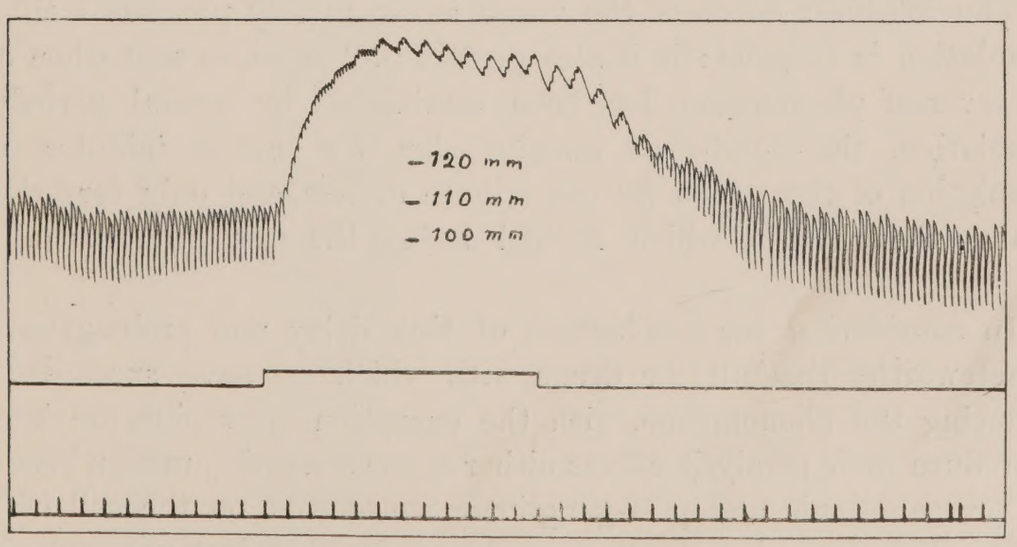

Fig. 3. Cat. Paraldehyde. Stimulation of the vagus after 60 mgm. of tropine. Coil 10 cm. Complete reversal.

following stimulation of the right, produces an acceleration quite similar to that produced by renewed stimulation of the right. (See Fig. 8.) It will be convenient to divide the phenomenon for consideration into two phases, and to examine separately the cause of the after-slowing, and that of the re-acceleration on renewal of the stimulation.

I. *The slowing of the heart-beat after cessation of the stimulus.*

We think that there can be little doubt that this phase of the effect is due to delayed inhibitor action of the vagus. If atropine is injected during a period of slowing the beat is promptly accelerated to a rate equal to or greater than that which obtained before the first stimulation period of the series. Apart from its time-relations this phase has all the characters of vagus-inhibition. By the use of Lehndorff's double cardiometer, which records the volume-change of auricles and ventricles separately, we found that the retardation affected the former concurrently with the latter (Fig. 4). The same observation can be made by simply watching the pulse in the exposed jugular vein. All the chambers of the heart are, therefore, affected as in normal vagus inhibition. All degrees of this delayed and prolonged inhibition were observed in different experiments: in one the inhibition was complete, the beat of the ventricle ceasing suddenly when stimulation of the vagus was stopped, and reappearing only after several seconds (Fig. 5) unless stimulation of the vagus was meanwhile renewed, in which case the beat began again promptly and rapidly rose to the rate obtaining during the previous period of stimulation.

The inhibitor effect of the vagus is not merely postponed till the stimulation is stopped: it is also greatly prolonged, so that when once the reversal phenomenon has been established by several periods of stimulation, the heart-beat remains slow for several minutes after stimulation of the vagus for one minute or less, and only very slowly regains the rhythm which it had before the first stimulus of the series.

In considering the mechanism of this delay and prolongation, it is noteworthy that all the drugs with which we have succeeded in producing the phenomenon, with the exception of tropine, are known to produce their paralytic effects either at the synaptic junction between the pre-ganglionic and post-ganglionic neurone or on the cell of the latter. The fact that tropine produces, in this instance, an effect similar to that of nicotine, curare and hordenine methiodide, may

possibly indicate that it, too, has a weak paralytic action of the nicotine type : it has been shown by several observers to have no true atropine action. It seems reasonable to suppose, therefore, that one phase of the phenomenon is due to delay and prolongation of the normal inhibitor effect of the vagus by partial obstruction to the passage of

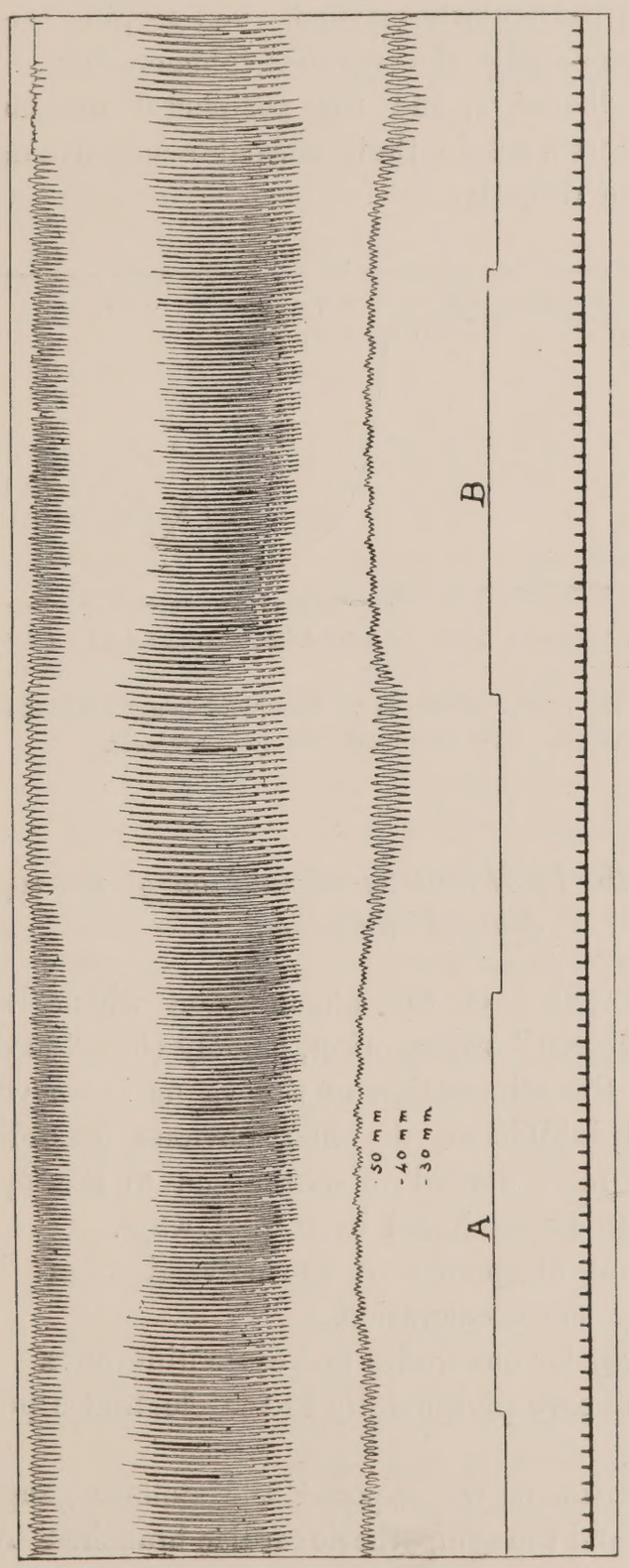

Fig. 4. Cat. Paraldehyde. Lehndorff's double cardiometer. Lines of tracing from above downwards show auricle volume, ventricle volume, carotid blood-pressure, signal key, time in seconds. 2 mgm. of nicotine. At A and B stimulation of right vagus : coil at 15 cm.

impulses across the synaptic junction. This is in accordance with the accepted fact that changes in the time-relations of nerve-impulses occur normally only at synaptic junctions. It is further supported by the observation that, when the reversal phenomenon has been fully developed, pilocarpine still produces its normal cardio-inhibitor effect, as does also a further and larger injection of nicotine. This inhibitor effect of nicotine was particularly marked when tropine was the drug primarily used in producing the reversal phenomenon. The post-ganglionic neurones, therefore, and the peripheral mechanism with which they are connected, retain their normal responsiveness to such stimuli as affect them directly.

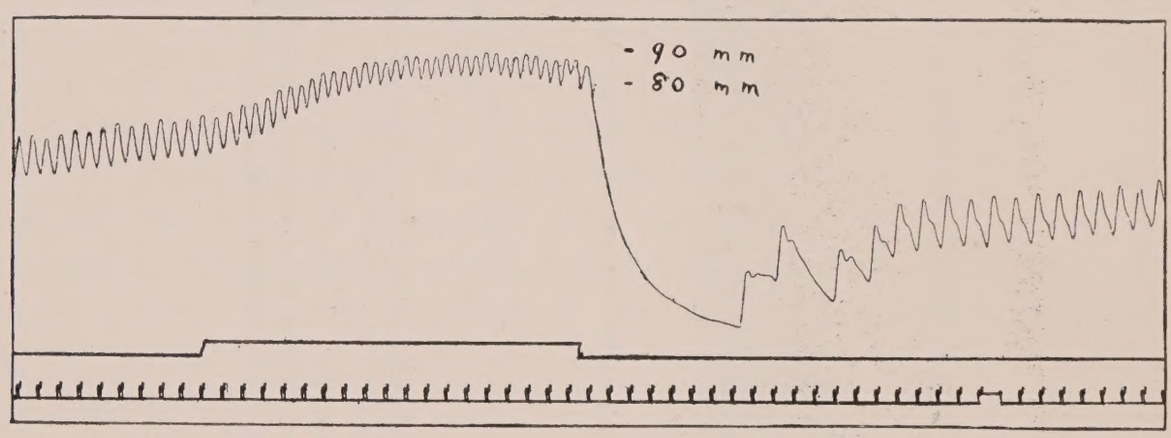

Fig. 5. Cat. Urethane. 150 mgm. of tropine. Complete delayed inhibition succeeding stimulation of the left vagus. Coil at 12 cm. Time in seconds.

II. *Acceleration of the heart-beat by stimulation of the vagus during delayed inhibition.*

Turning to that phase of the phenomenon which leads us to describe it as a "reversed" vagus effect, namely the re-acceleration of the heart-beat when the stimulation of the vagus is renewed during a period of delayed inhibition, the first obvious possibility to be considered is that of mere spread of current on to the sympathetic accelerators. This can be excluded in several ways.

(1) Stimulation at all points of the cervical vagus is equally effective in producing the acceleration.

(2) The use of tripolar electrodes makes no difference.

(3) Pinching the nerve peripherally to the electrodes prevents the acceleration.

(4) In one experiment we exposed the stellate ganglion with its rami, the annulus, and the sympathetic cardiac branches. A stimulus,

which, applied to the sympathetic accelerators at any point, during a period of delayed inhibition, produced no effect or a barely perceptible acceleration, caused marked acceleration when applied to the vagus of the same side. (Fig. 6.)

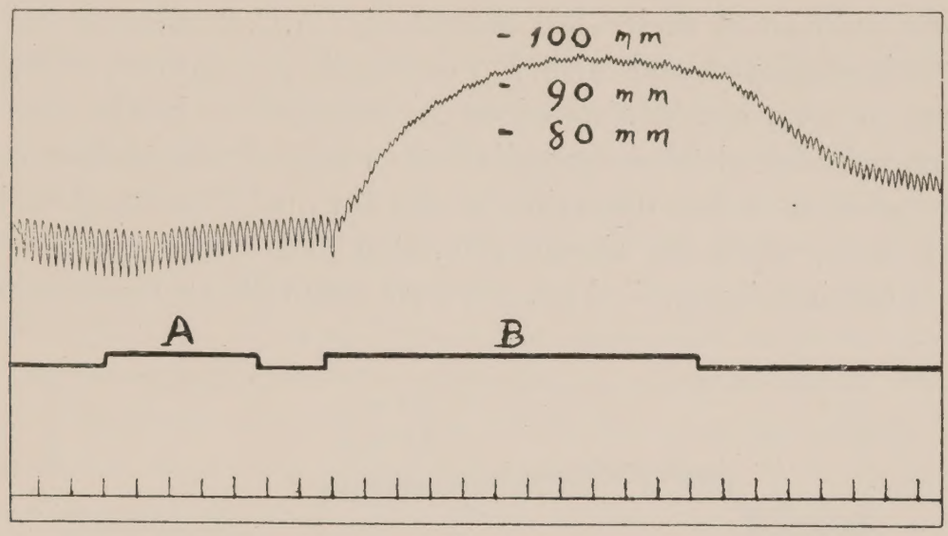

Fig. 6. From same experiment as Fig. 1.

A. Stimulation of right sympathetic accelerators, coil at 16 cm.
B. Stimulation of right vagus, coil at 16 cm.

Current escape is not, therefore, a possible explanation.

A second possible explanation of the abnormal accelerator effect is that it is due to sympathetic post-ganglionic fibres running in the vagus trunk, their effect being normally overpowered, when the vagus is stimulated, by the intrinsic inhibitor fibres. The close connexion of the vagus with the annulus in the cat, especially on the right side (cf. figure by Boehm [6]), and the fact that strands of non-medullated fibres can be traced running between them when they are teased apart, lend support to the suggestion that fibres from the stellate or inferior cervical ganglion may run up for some distance in the vagus and loop back to their distribution. We have already used the possibility that such fibres exist to explain such results as those of Rutherford, Schiff, Boehm and Arloing. The fact that some of these observers obtained more marked accelerator effects with the right than the left vagus is in favour of this explanation of their results. Most of the reasons which exclude current escape may be urged, however, against the attribution of our phenomenon to the presence of such fibres. But for the complete exclusion of this possibility, and of the further possibility that accelerator fibres may arise in the superior cervical

ganglion and join the vagus therefrom[1], degeneration experiments are necessary. Such were therefore undertaken. In a series of experiments the superior cervical ganglion, the inferior cervical ganglion, and the stellate ganglion were removed in separate cats, and in addition all three were removed from each of two cats. The operation was in all cases performed under full anæsthesia, A.C.E. mixture being the anæsthetic employed, and with full antiseptic precautions. The operation was in every case performed on the right side. In the case of the two cats in which all three ganglia were removed the stellate ganglion was removed at a first operation by Anderson's[7] method, and about a week later, when the shoulder incision had healed completely, the superior and inferior cervical ganglia were removed at a second operation.

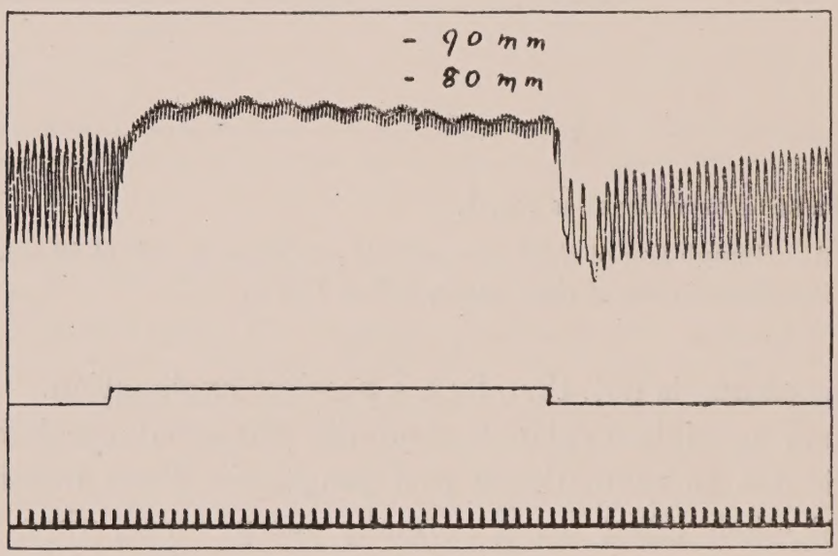

Fig. 7. Cat. Paraldehyde. Superior and inferior (middle) cervical and stellate ganglia removed by operation 12 days previously. 1 mgm. nicotine. Stimulation of the right vagus: coil at 15 cm.

Healing occurred in all cases by first intention. Degeneration was allowed to proceed for varying periods up to 31 days, this latter period being allowed in one of the experiments with extirpation of all three ganglia. At the end of the period of degeneration the animal was anæsthetised in the usual manner with urethane or paraldehyde and the right vagus tested for the production of the phenomenon of reversed action. In all cases it was produced in quite a normal manner. Fig. 7 shows the reversed action of the right vagus in a cat from which all three ganglia were extirpated on the right side, degeneration being allowed

[1] Langley (Schäfer's *Text-book of Physiology*, Vol. II. p. 663) suggests the presence of such fibres in the vagus in explanation of Rutherford and Schiff's results.

for 12 days subsequent to the second operation. It is clear, therefore, that fibres of the true sympathetic system can play no part in the production of the effect.

The accelerator effect being thus undoubtedly due to fibres belonging to the vagus itself, we have a further choice of possibilities to consider.

In the first place, it is conceivable that an actual reversal of function takes place under the influence of the drug, fibres which are normally inhibitor becoming accelerator under the abnormal conditions of the experiments. On such a view the primary effect would be the delay and prolongation of inhibition. During this period of delayed inhibition the post-ganglionic inhibitor neurone must be in a state of mild continuous activity. It is at least conceivable that impulses reaching it from the pre-ganglionic fibre under these conditions might interfere with the continuous excitation, the result being escape of the heart from inhibition while the stimulus lasts. There are, however, several objections to such a view. The effect of the first stimulation after administration of the paralytic drug is frequently a perceptible acceleration while the stimulus lasts, though this is naturally not so marked by contrast as the acceleration produced by subsequent stimulation during periods of delayed inhibition. Secondly, the acceleration is not a mere removal of the delayed inhibition: when the phenomenon has been well established by successive periods of stimulation, the acceleration frequently carries the rate above that which obtained subsequent to the administration of the drug, but prior to the first period of stimulation. Thirdly, a condition of not quite complete reversal is frequently met in which the first effect of the onset of each period of stimulation is to cause a further inhibition, lasting for a second or two, and followed by acceleration which then lasts till the stimulation is stopped. None of these features of the phenomenon can easily be reconciled with the hypothesis of interference.

The other, and on the whole the more obvious explanation, is that the vagus contains, in addition to its normally predominant inhibitor fibres, others which produce acceleration of the heart-beat. The function of the paralyzing drug in this case would be discriminating, the accelerator action being revealed by depression of the inhibitor effect which normally masks it.

If we accept the view that the vagus contains cardio-accelerator fibres, we could suppose these to be (*a*) pre-ganglionic autonomic fibres

like the inhibitors; (*b*) post-ganglionic autonomic fibres, having their cell-station in the jugular ganglion or the ganglion of the trunk; (*c*) fibres which are normally sensory in function, but capable, on artificial excitation, of producing acceleration by antidromic action, the effect being analogous to the vaso-dilatation caused by stimulating dorsal spinal roots (Stricker[8], Bayliss[9]). We have unfortunately not found it possible to discriminate experimentally between these possibilities. With none of the various methods, which have been used in other directions to reveal admixtures of fibres having different

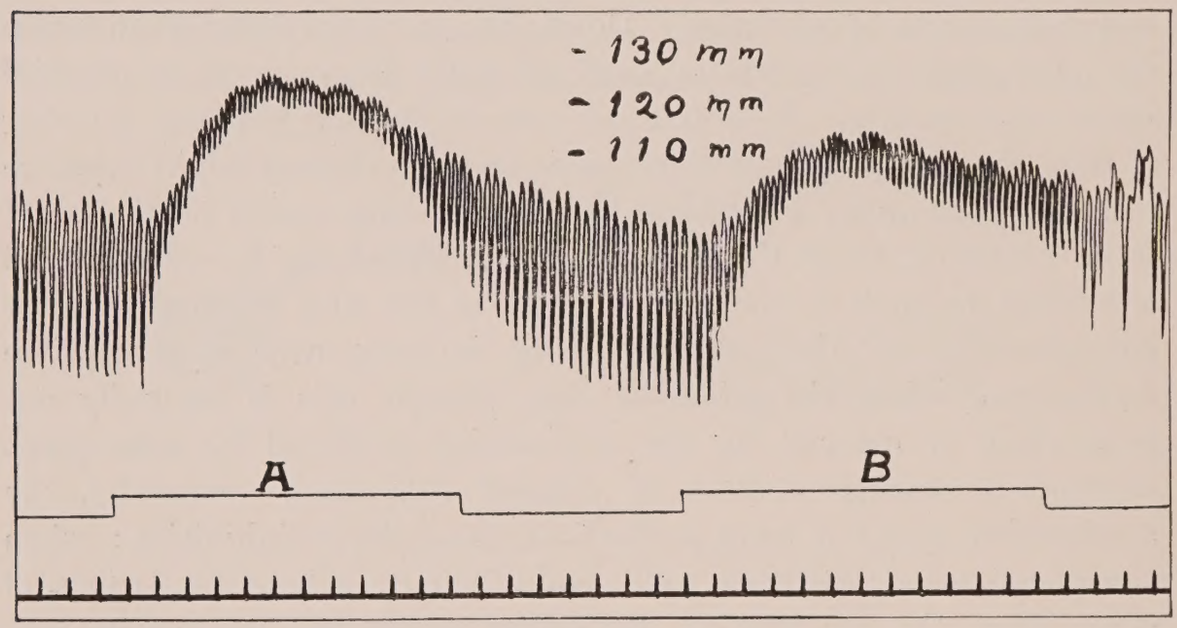

Fig. 8. Cat. Paraldehyde. Right vagus cut 2 days previously.
A. Stimulation of right vagus, coil at 12 cm.
B. ,, left ,, ,, ,,
Time in seconds.

functions and origins, have we succeeded in producing acceleration with the vagus, in the absence of the paralyzing drug. Slow rhythmical stimulation by induction shocks, repeated make-and-break of a constant current, mechanical excitation, have, in our experience, produced either no effect or inhibition. Nor could we separate the two effects by fractional degeneration. In anæsthetised cats one vagus was cut with aseptic precautions, and degeneration allowed for varying periods, at the end of which the animals were again anæsthetised with paraldehyde and the production of the reversal phenomenon investigated. We found that the accelerator action, under the conditions producing reversed action of the other vagus, persisted in the divided nerve as long as the normal inhibitor action and no longer. After two

days' degeneration the complete phenomenon was obtained, delayed inhibition and re-acceleration during the succeeding stimulus being produced in the characteristic manner. (Fig. 8.) In another case, in which degeneration had proceeded for four days, stimulation of the degenerated nerve produced neither inhibition nor acceleration, before or after the injection of a small dose of nicotine: nor did stimulation of the divided nerve produce any trace of re-acceleration when applied during a period of delayed inhibition, succeeding stimulation of the sound nerve of the opposite side (Fig. 9).

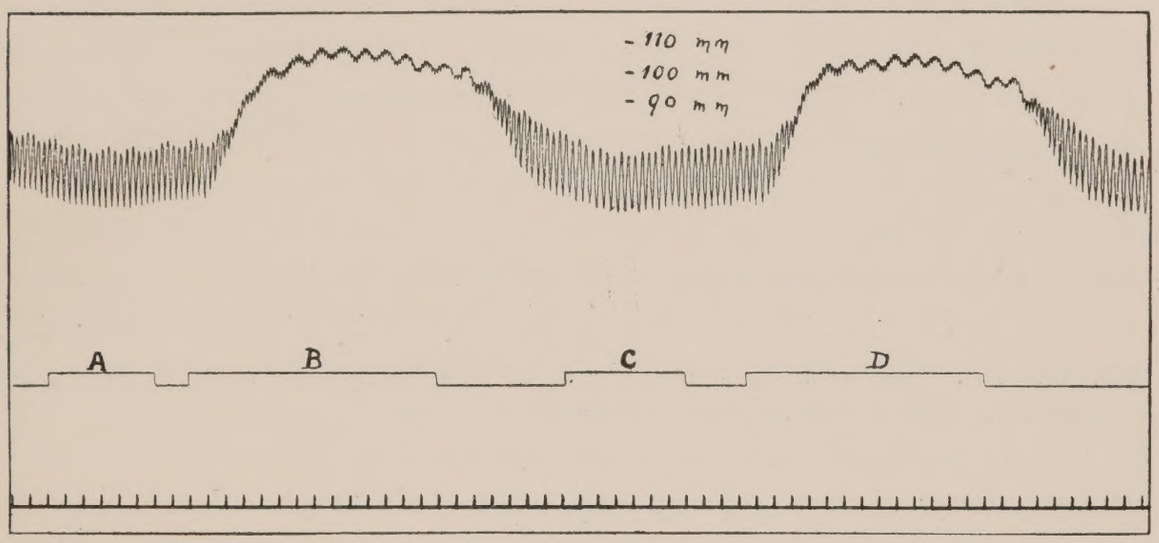

Fig. 9. Cat. Paraldehyde. Right vagus cut 4 days previously.

A. Stimulation of right vagus, coil at 8 cm.
B. ,, left ,, ,, 11 cm.
C. ,, right ,, ,, 5 cm.
D. ,, left ,, ,, 11 cm.

An exceptional result, to which reference has already been made, may conveniently be mentioned here. In one cat only the right vagus, 19 days after section, produced on stimulation a marked acceleration of the heart-beat (Fig. 10). Unfortunately the nerve was not cut high in the neck; but even with the comparatively short peripheral portion available it was easy to satisfy ourselves that the acceleration effect increased as the electrodes were shifted towards the heart. The acceleration, moreover, was only obtained from any portion of the nerve with comparatively strong stimulating shocks. (Secondary coil at 10 to 8 cm. from the primary.) We attribute this result, therefore, like those of Schiff and Arloing, to the presence of recurrent fibres from the sympathetic ganglia. No sound medullated fibres could be

detected microscopically in the nerve after fixation in osmic acid and careful teasing.

On one other point we obtained decisive evidence. If the accelerator fibres are post-ganglionic or sensory their cell-station must be in the jugular ganglion. The vagus was cut in a cat under A.C.E. and with aseptic precautions, just above the ganglion of the trunk. After degeneration for ten days it was tested as usual, the cat being under urethane. No inhibitor or accelerator effect was obtained. This almost suffices for the exclusion of an antidromic effect as the cause of the acceleration: for even if the possibility be admitted that a few

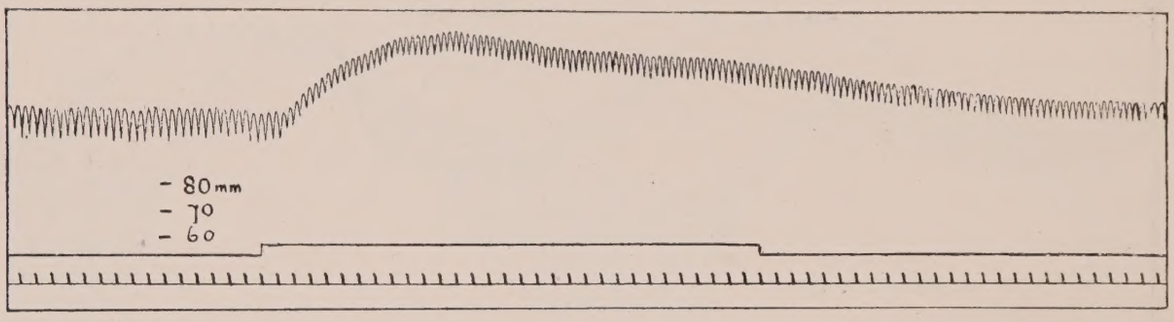

Fig. 10. Cat. Urethane. Exceptional effect of stimulating right vagus 19 days after section. Coil at 8 cm. Time in seconds.

sensory fibres from the heart may have their trophic centre in the jugular ganglion[1], it seems most improbable that these alone should be capable of conveying antidromic impulses causing acceleration, the fibres connected with cells in the ganglion of the trunk being ineffective in this direction. The possibility remains that the jugular ganglion may contain cells of the autonomic type, giving origin to post-ganglionic accelerator fibres. This could only be excluded by intracranial section of the vagus roots, with subsequent degeneration. We have been unable to devise a method for performing this operation in the cat.

Keeping this in view as a possibility, it seems to us, nevertheless, that the balance of probability is in favour of the presence in the vagus of pre-ganglionic fibres, stimulation of which causes acceleration of the heart. The chief difficulty in the way of this conception is that it postulates a discrimination by the paralyzing drug between cells or synapses concerned with inhibition and acceleration respectively. Such discrimination is no new conception in the case of more peripherally

[1] According to Langley all the sensory fibres found in the vagus below the ganglion of the trunk probably have their trophic centre in that ganglion. (This *Journal*, XXVII. p. 230. 1901.)

acting alkaloids. Langley[10] showed, for example, that atropine abolished the motor before the inhibitor effect of the vagus on the rabbit's cardiac sphincter. An abolition of motor effects of sympathetic nerves and of adrenine by ergotoxine, without noticeable impairment of their inhibitor effects, has been described by Dale[11]. Some of the effects produced by small doses of ergotoxine are, indeed, strongly reminiscent of the reversal of the vagus effect on the heart. Stimulation of the splanchnic nerves, for example, after a small dose of ergotoxine, frequently causes a small initial rise of arterial blood-pressure, giving way to a fall which persists while the stimulation continues, to be succeeded by a further prolonged rise when the stimulation ceases. These effects have been generally accepted as indicating a discrimination by the paralyzing alkaloid between motor and inhibitor elements in the peripheral receptive mechanism. Such discrimination in the case of ganglion-cells has, to our knowledge, not been described hitherto, but it seems to us to present no greater inherent difficulty of conception than the analogous process at the periphery.

The view that the accelerator effect is due to the presence of normally masked accelerator fibres also renders easier the explanation of that phase of the phenomenon which we have described as delayed inhibition. It is a common effect of a paralyzing drug, in doses short of those necessary for complete annulment of an effect, to produce a reduction of intensity associated with increased persistence. If we suppose that the effect of the inhibitor neurones is thus altered, it is easy to understand why it should be overcome, during the application of the stimulus, by an accelerator effect which is not similarly depressed and prolonged. When the stimulation ceases the accelerator effect rapidly passes off and allows the prolonged, weak inhibitor effect to emerge.

The fact that the phenomenon has been observed only in such typically carnivorous species as the cat and the ferret is in favour of the theory of selective paralysis as opposed to that of reversal of function. It was in these types that the most striking instances were observed of mixed motor and inhibitor function in sympathetic nerves, as revealed by ergotoxine. It was in the cat, again, that Brodie and Dixon[12] observed a mixed motor and inhibitor action of the vagus on the muscles of the bronchioles. On the whole, therefore, while realising that the somewhat complicated conditions, needed for the production of the phenomenon in its most striking form, make caution desirable in

drawing conclusions, we are of opinion that the balance of probability is in favour of the view which regards the vagus supply to the cat's heart as of mixed function, though predominantly inhibitor under normal conditions. Such an admixture of accelerator fibres would help to explain the readiness with which the cat's heart escapes from vagus inhibition.

Note on an analogous phenomenon observed on the cat's pupil.

We have in several experiments observed a modified response of the cat's pupil and nictitating membrane to stimulation of the cervical sympathetic, which we regard as analogous to the reversed vagus effect on the heart, described above. The cats have usually been anæsthetised with paraldehyde, in the manner already described, but this does not seem to be a point of such importance in this instance, as the phenomenon can be observed in cats under chloroform or ether.

The cervical sympathetic nerve on either side is first exposed and faradised in the usual manner, the weakest stimulus which will just produce maximal dilatation of the pupil being thus determined. Nicotine in 0·1 % solution in saline is then painted on the superior cervical ganglion and the cervical sympathetic stimulated from time to time, the coil being approximated as the effect becomes weaker. If the dilator effect is not abolished by 0·1 % nicotine, stronger solutions up to 0·5 % are employed. Ultimately the pupil does not respond to any strength of stimulus. The animal is now left for a time and the stimulation again applied, and repeated at intervals. In some cases nothing occurs until ultimately a weak normal dilator effect reappears. In most cases, however, after a short time, a curious delayed effect is observed. A slight and rapid dilatation may occur at the onset of the stimulus, but this quickly passes off and the pupil remains in its semi-contracted condition while the stimulus continues. As soon as it ceases slow dilatation of the pupil and retraction of the nictitating membrane appear, and the dilated condition persists for some seconds. If now, during this period of delayed dilatation, the stimulus is renewed, a slight but quite distinct constriction of the pupil and advance of the nictitating membrane occurs and persists while the stimulation lasts, again giving way to delayed dilatation and retraction when the stimulation stops. Quite similar results were obtained by painting 0·2 % hordenine methiodide on the superior cervical ganglion. When once the

phenomenon has been produced it is not easily abolished by further painting of the ganglion with nicotine.

We have made no analysis of the effect, except to exclude the participation of possible pre-ganglion fibres running through the superior cervical ganglion and making connexion with cells of the ciliary ganglion. Injection of 1 % nicotine into the orbit intensifies the effect if it alters it at all.

The effect is a small one and needs close watching for its observation, but, in a favourable case, the edge of the iris may be seen to move 1—2 mm., while the movement of the nictitating membrane, though less constant, is sometimes the most prominent feature in the effect.

It seems to us almost certain that this phenomenon and the heart-effect with which this paper is principally concerned must be explained along the same lines.

A reversal of the action of the pelvic nerve on the cat's bladder, produced by curare, was demonstrated by Prof. Langley[13] to the Physiological Society on July 9th. In this case, again, the order of events, viz. a preliminary contraction at the onset of the stimulation, followed by relaxation while the stimulation continued, and a further, much larger and more persistent contraction following the cessation of the stimulus, is strongly suggestive of analogy with the reversed vagus effect on the heart. Langley regards his observation as indicating the presence in the cat's pelvic nerve of an admixture of inhibitor fibres for the bladder.

Summary.

A reversal of the effect of the vagus on the heart of the cat is described, by which acceleration of the rhythm during electrical stimulation of the nerve is produced with inhibition following the cessation of the stimulus. It is uncertain whether the phenomenon is due to actual reversal of the function of normally inhibitor fibres, or to the presence in the vagus of normally masked accelerator fibres. The latter seems the more probable view.

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